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Case Report

Atypical presentation of leprosy in HIV

AK Manjare, SA Tambe, MM Phiske, HR Jerajani

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Atypical presentations can be expected when leprosy, a mycobacterial disease is associated with HIV. We report a case of a 28 year old male driver with a high risk behavior, who came for evaluation of hypoaesthetic, scaly erythematous plaques over face, trunk, upper extremity; verrucous lesions over elbows and necrotic lesions over the neck and lower extremities since 6 months. No other systemic complaints were present. Nerve examination showed grossly thickened left greater auricular nerve and cord like thickening of bilateral ulnar and lateral popliteal nerves. His investigations revealed anemia, a reactive ELISA for HIV-1 and CD4 of 400 cell/cmm. Ultrasonography of the thickened nerves revealed an abscess in the left ulnar nerve whereas the left greater auricular nerve showed neuritis. Histopathology from an erythematous plaque was suggestive of borderline tuberculoid leprosy in reaction. Final diagnosis was borderline tuberculoid leprosy in type 1 reaction with atypical and varied morphology in an immunocompromised male with neuritis of the left greater auricular nerve, a silent left ulnar nerve abscess with early left ulnar nerve palsy. Our case highlights the atypical morphology of leprosy lesions and the unexpected protective cellular response as suggested by formation of nerve abscess in a HIV positive patient.

Key words : Leprosy, Human immunodeficiency virus, Nerve abscess, Ultrasonography of nerve

Introduction

Early in the HIV epidemic, it was feared that the disease would undermine leprosy control as has occurred with tuberculosis. It was predicted that patients with leprosy and HIV co-infection would have an increased risk of lepromatous disease and a faster clinical evolution and that the leprosy would be more difficult to treat. None of these concerns have materialised and the interaction between HIV and *Mycobacterium leprae* seems to be far more subtle than that between HIV and tuberculosis (Ustanowski et al 2006).

Case Report

A 28 year old male, driver by occupation, presented with multiple, scaly, reddish lesions over bilateral upper extremities, face, neck and back along with crusted lesions over the lower extremities since 5-6 months. He complained of decrease in sensations over the lesions but there was no history of any constitutional symptoms like fever and malaise. He did not complain of tingling numbness, cotton wool sensations while walking, radiating pain along the extremities, muscle weakness, joint pain or difficulty in daily activities. There was no history of unnoticed

AK Manjare, MBBS, Post-Graduate Resident
SA Tambe, MBBS, Post-Graduate Resident
MM Phiske, MD, Assistant Professor
HR Jerajani, MD, Professor and Head of the Department
Department of Dermatology, Lokmanya Tilak Municipal Medical College and General Hospital,
Sion, Mumbai-400 022, Maharashtra, India
Correspondence to: AK Manjare **Email :** ashwinimanjare@yahoo.co.in

trauma or non healing wounds. On further enquiry, patient confessed to high risk behavior. His past medical history was unremarkable. Family history was not contributory. He denied any addictions. His vital parameters were normal and there was no evidence of any pallor, icterus, clubbing, lymphadenopathy or oedema. Cutaneous examination revealed well defined, irregular, erythematous, scaly plaques with overlying shiny surface measuring approximately 7x5 cms and 4x3 cms over the dorsae of the right and left hand (Figure 1) respectively with hollowing of interossial spaces, more evident on the left hand. Irregular, grayish, verrucous plaques measuring 4x3 cms and 3x2 cms were present on the left and right elbows respectively with central clearing and atrophy (Figure 2). Necrotic plaques were present over the neck and the pinna of the left ear. The back showed evidence of annular plaques with hyperpigmented scales. The sensations of touch, pain and temperature were decreased over the lesions and also in the glove and stocking pattern over the extremities. Corneal and conjunctival sensations were intact. Motor examination revealed a normal tone and power in all muscles of the extremities expect a grade IV power in the muscles of the left upper extremity. There was evidence of flattening of the thenar and hypothenar eminences. Nerve examination revealed gross visible thickening of the left greater auricular nerve (Figure 3) and bilaterally thickened and cord like ulnar and popliteal nerves. On palpation of the nerves, there was no evidence of any tenderness, beading or soft swelling. His routine haemogram and chemistry profile was unremarkable except for mild anaemia. ELISA for HIV 1 and 2 done due to his high risk behaviour was reactive for HIV 1 with a CD4 count of 400 cells/cumm. VDRL was non-reactive. Histopathology from an erythematous plaque revealed hyperkeratosis overlying an atrophic epidermis with upper dermal oedema and dense lichenoid infiltrate composed of

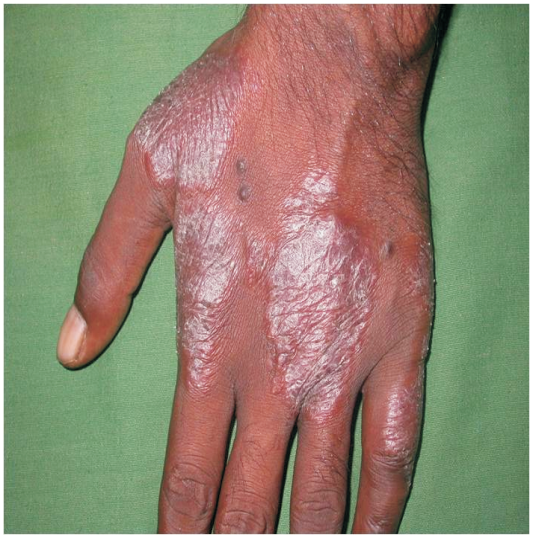

Figure 1: Well defined, irregular, erythematous, scaly plaques with overlying shiny surface measuring approximately 7x5 cms and 4x3 cms over the dorsae of the right and left hand respectively with hollowing of interossial spaces, more evident on the left hand.

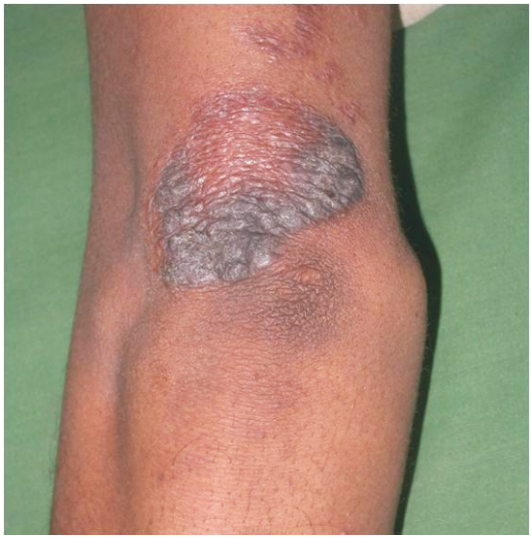

Figure 2: Irregular, grayish, verrucous plaques measuring 4x3 cms and 3x2 cms were present on the left and right elbows respectively with central clearing and atrophy.

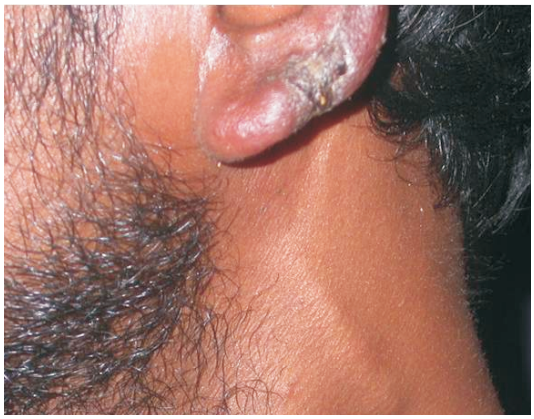

Figure 3 : Gross visibly thickened left greater auricular nerve.

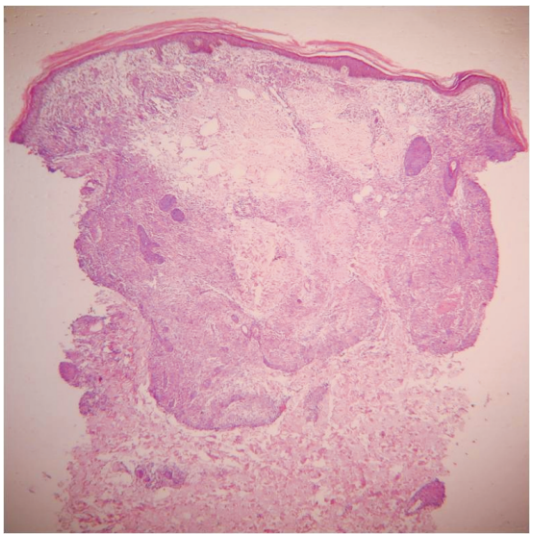

Figure 4 : 4 X; H&E. Presence of hyperkeratosis overlying an atrophic epidermis with upper dermal oedema and dense lichenoid infiltrate composed of lymphocytes and occasional giant cells. Multiple granulomas composed of epithelioid cells and lymphocytes were seen to occupy the entire dermis.

lymphocytes and occasional giant cells. Multiple granulomas composed of epithelioid cells and lymphocytes were seen to occupy the entire dermis (Figure 4) with infiltration of the nerves in

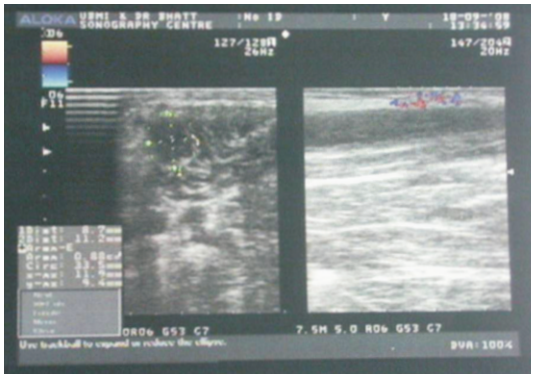

Figure 5 : Ultrasonography of the left ulnar nerve (transverse and longitudinal view). Anechoic nerve and hyperechoic bands, suggestive of an abscess seen on transverse view.

the subcutaneous tissue. Chest radiograph was unremarkable. USG abdomen revealed splenic microabscesses. As part of an ongoing study at our institute Ultrasonography of the thickened nerves was performed which on transverse scan surprisingly revealed an anechoic left ulnar nerve with hyperechoic bands, suggestive of an abscess (Figure 5) and presence of oedema in the left greater auricular nerve suggestive of neuritis. Clinicopathological correlation clinched the final diagnosis of untreated borderline tuberculoid leprosy in type 1 reaction with atypical and varied morphology in an immunocompromised male with neuritis of the left greater auricular nerve, a silent left ulnar nerve abscess with early left ulnar nerve palsy. He was started on MDT-MB with oral prednisolone in reaction dose with subsequent tapering on improvement.

Discussion

In the Era of HIV pandemic it was expected that the prevalence of leprosy would increase in HIV positive patients with a change of clinical spectrum towards the lepromatous pole, this was based on the fact that the type of leprosy in an individual depends on the cellular immune response which is depleted in HIV. But contrary to this belief, HIV does not affect the migration of *M. leprae* specific CD4+ T cells to the lesion site

or their response to the antigen (Sharma and Malhotra 2008) .

There are very few case reports of leprosy in association with HIV (Schettine et al 1996, Goodless et al 1994) available in the literature. Spectrum of skin lesions ranging from hypo-pigmented tuberculoid lesions to nodular lepromatous lesions have been described in HIV infected patients (Singhal 2010). Few atypical and rare skin manifestations of leprosy have been reported in association with HIV.

A case of borderline tuberculoid leprosy in a HIV-positive patient who developed a marked reversal reaction was reported and it was noted that HIV co-infection seemed to be associated with an increased rate of reactional states and more severe cases of neuritis. Atypical and rare skin manifestations, such as verrucous lesions and ulcers, appeared after highly active antiretroviral therapy which resulted in increased CD4+ T-lymphocyte count and drop in viral load.

Contrary to the above case, antiretroviral was not initiated in our case, yet patient had atypical lesions, was in reaction and had neuritis and a silent nerve abscess.

Worsening of nerve damage might be expected in patients with dual infection as HIV infection may alter the immune response in nerves to *M. leprae.* HIV is itself neuropathic; so, it could also act synergistically. However, conclusive evidence to support this hypothesis is lacking (Singhal 2010).

Immunologically driven inflammation is also responsible for much of the clinically apparent nerve injury. Nerve function impairment is more rapid and severe in patients with aggressive cellular immune response i.e. in tuberculoid disease and during reactional states, especially type 1 reaction. As HIV principally affects cell mediated responses, these pathogens may also have potentially interesting immunologic interactions in the human host. Unlike tuberculosis, where HIV infection affects granuloma formation depending on the degree of immune suppression as reflected by blood CD4+ counts, host granulomatous response is preserved among individuals co-infected with HIV and leprosy (Singhal 2010).

Bacterial parasitization of peripheral nerves is a unique feature that is characteristic of leprosy. In majority of the immunocompetent patients, the resulting neural lesion remains as a granuloma but in a few cases the granuloma may soften and develop into an 'abscess.' Progression to abscess formation is most commonly seen in patients with tuberculoid leprosy. Rarely, however, nerve abscess may also develop in other types of leprosy (Kumar et al 1997).

The usual habitat for *M. leprae* in the nerve is the Schwann cell but occasionally the ensheathed axon becomes involved. The Schwann cells assume a phagocytic function and evolve into macrophages or epitheloid cells resulting in the formation of a granuloma. Invasion of the endoneurium may follow and the whole endoneurial zone may appear to be occupied by epitheloid cells with or without the presence of bacilli. Caseation may occur in microscopic foci within the granulomas or areas of necrosis may coalesce, forming a cold abscess particularly when the immunity is high. Cold abscesses occur more frequently in the tuberculoid form especially in India (Singh and Ojha 1969, Char and Cross 1986).

Our case is one of a kind as a nerve abscess which indicates a good immunity was present in an immunocompromised male. To our knowledge, this is the first case report of such an association. In our case, high-resolution ultrasound helped us in unearthing a silent nerve abscess. Thus, highlighting the long term utility of this non-invasive investigational modality in early detection of silent neuritis or nerve abscess in cases of leprosy (Taneja et al 1992), thereby helping prevent deformities and disabilities

Conclusion

Our case highlights the atypical morphology of leprosy lesions, the unexpected protective cellular response as suggested by formation of nerve abscess in a HIV positive patient and a

clinically silent nerve abscess which was diagnosed on high resolution ultrasound of the nerve.

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